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CASE REPORTS

Recurrent Ventricular Tachycardia

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Ventricular tachycardia is a dangerous form of arrhythmia, and is usually a manifestation of serious heart disease, either acute ischaemia, severe heart failure, myocarditis, or sometimes drug intoxication, especially digitalis. Treatment is only rarely based on aetiology. The most effective therapy has been found to be electrical conversion (Lown *et al.*, 1963; Castellanos, Lemberg, and Fonseca, 1965; Waris, Kreus, and Salokannel, 1967). However, defibrillation does not prevent relapses, and therefore it is often compulsory to repeat cardioversions and to use antiarrhythmic agents to retain sinus rhythm. The purpose of the present paper is to report a case of recurrent ventricular tachycardia, and to show that the key problem is how to prevent new attacks after successful electrical conversion.

Case Report

A shopkeeper, aged 56, was first examined in 1964 in the Department of Medicine, University of Oulu. Two years earlier he had had a myocardial infarction, but he recovered and was able to resume work. He was admitted to our hospital for ventricular tachycardia of three days' duration despite treatment with quinidine and procainamide. On admission he was in a poor condition, being cyanotic, hypotensive, and in heart failure. The electrocardiogram revealed ventricular tachycardia, with a rate of 180. He was given quinidine sulphate (2·4 g. a day) and four doses of procainamide, 1000 mg. each, intramuscularly or intravenously. Since tachycardia persisted for 10 days after admission, a non-synchronized direct current countershock was given. This was the first defibrillation in our hospital. A single 100 watt-second shock converted the heart to sinus rhythm. The electrocardiogram revealed sinus rhythm, the characteristics of inferior wall infarction, and ischaemic anterior wall lesions. After defibrillation he was given digoxin 0·25 mg. twice daily, quinidine bisulphate 0·4 gr. twice daily, and prednisolone 5 mg. twice daily. Twelve days after defibrillation he had the first relapse (Fig.). Intravenous procainamide and carotid sinus massage failed, but the first shock (100 watt-seconds) again restored sinus rhythm. During the following five days there were two relapses, both converted to sinus rhythm by defibrillation.

There were nine more attacks of ventricular tachycardia during the next 11 months, and each one was converted to sinus rhythm by a single 100 watt-second D-C shock. During the next year the patient had one attack in January and another in November. The following year the first relapse occurred in January. Apart from a few weeks in the summer, the patient was then permanently in hospital because of recurring attacks of ventricular tachycardia and poor condition. He died at the end of October 1966.

In all, this patient was defibrillated for 47 attacks of ventricular tachycardia. The first shock of 100 watt-seconds resulted in sinus rhythm in 40 attacks. On one occasion a second and on another a third 100 watt-second discharge converted ventricular tachycardia to sinus rhythm. Four times a 200 watt-second discharge was required for a successful result. Only once were five shocks, an energy level up to 400 watt-seconds, needed before sinus rhythm returned.

Besides countershocks many attempts at conversion were made with antiarrhythmic drugs. Procainamide given intravenously or intramuscularly was sometimes successful in converting the heart to sinus rhythm, whereas quinidine sulphate orally in a dose of 2·4 g. (the maximum dose tolerated by the patient), propranolol intravenously or orally, xylocaine intravenously, ajmalin, antazoline chloride, and diphenylhydantoin failed. The same medicines, with the exception of xylocaine, were used to maintain the achieved sinus rhythm. The longest time between two attacks was about 10 months in 1965, when 50 mg. ajmalin was taken three times a day, prednisolone 10 mg. a day, and digoxin 0·25 mg. a day.

In July 1966, the patient had one or more attacks of ventricular tachycardia daily and in most cases countershock was needed for conversion.

In order to terminate tachycardia and control the heart rate, a bipolar electrode catheter was introduced

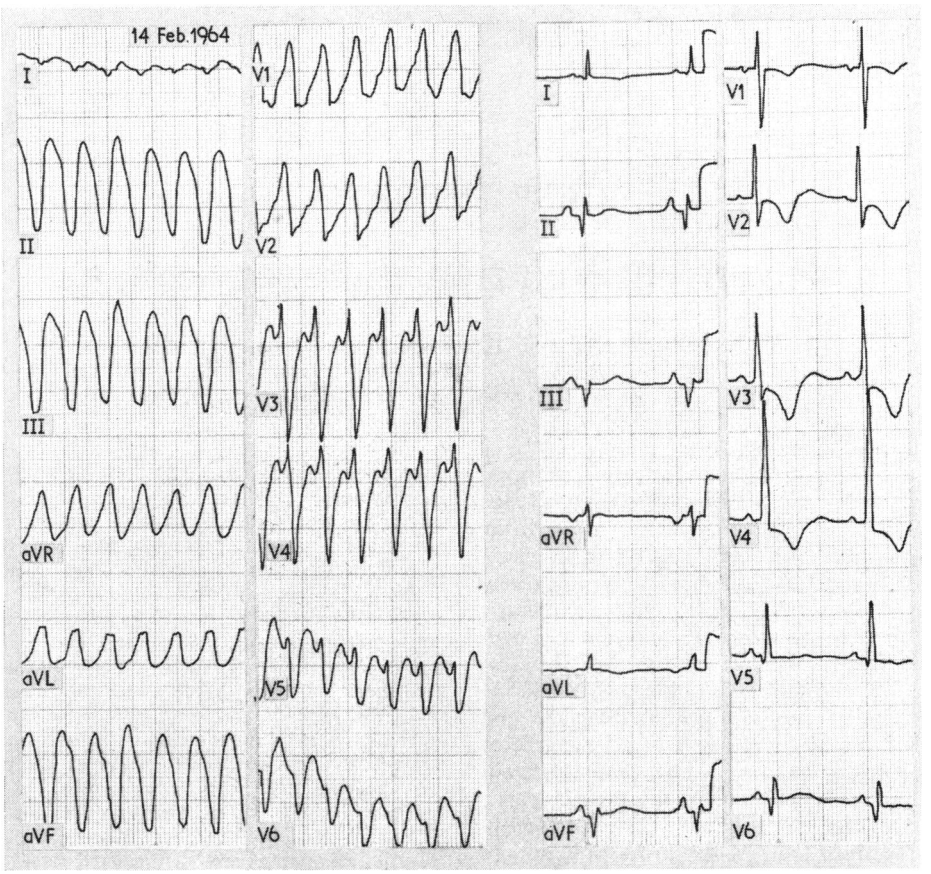

FIG.—Electrocardiogram shows ventricular tachycardia, with a rate of 200 and conversion of that to sinus rhythm by direct current defibrillation. See case report for details.

from an arm vein to the right ventricle. With single pulse pacing (Dressler, 1964; Härtel *et al.*, 1966; Robinson *et al.*, 1965; Sowton, Leatham, and Carson, 1964) an ectopic focus was suppressed, and sinus rhythm returned. In the oscilloscope both sinus rhythm and pacemaker impulses were seen. Despite different energy levels and pacemaker rates, sinus rhythm persisted only for a few minutes, after which ventricular tachycardia recurred. Once more the sinus rhythm returned by pacing, and now the pacemaker was switched off immediately. However, ventricular tachycardia reappeared after two hours, and subsequent conversion attempts with a pacemaker were unsuccessful, even with paired pulse pacing (Chardack, Gage, and Dean, 1964).

Sinus rhythm was achieved again by direct current defibrillation. Subsequently the patient was given procainamide orally—up to 6 g. a day—and was discharged from hospital. He was at home for two weeks, and was then readmitted in a very poor condition, severely dyspnoeic, hypotonic, with the heart decompensated. This time, however, the heart was in sinus rhythm except for some high nodal extrasystolic beats. The treatment was unsuccessful, and after six hours the patient died.

At necropsy the posterior wall of the whole heart was seen to be very thin, pale, and fibrotic, and no signs of fresh myocardial infarction were noted. Circumflex branches of the coronary arteries were slightly hypoplastic. The left circumflex branch was nearly completely occluded by atherosclerosis. There were also atheromatous patches in the right circumflex and left descending branches. A big mass of thrombus was found in the right atrium firmly attached to endocardium. The thrombus continued via the right ventricle to the main branches of the pulmonary artery.

Discussion

There is no doubt that direct current countershock is the most important improvement of recent years in the treatment of cardiac arrhythmias, especially in ventricular fibrillation and tachycardia. With defibrillation, ventricular tachycardia can

nowadays almost always be suppressed (Lown *et al.*, 1963; Castellanos *et al.*, 1965; Waris *et al.*, 1967). New antiarrhythmic agents such as propranolol, antazoline, diphenylhydantoin, and xylocaine have also been introduced (Harrison, Sprouse, and Morrow, 1963; Dreifus, Rabbino, and Watanabe, 1964; Harris, 1966; Stock, 1966), though procainamide is still the drug most used in medicinal treatment of ventricular tachycardia (Paul and Leigh, 1966). It is evident that we can today save the lives of many such patients with serious heart disease, who otherwise would have died. The problem is now how to prevent the recurrence of ventricular tachycardia after conversion to sinus rhythm. The present report is an illustration of this problem. We had to defibrillate this patient no fewer than 47 times for attacks of ventricular tachycardia during two and a half years. Usually the attacks were refractory to antiarrhythmic agents. Nearly all known antiarrhythmic agents were used in an effort to retain sinus rhythm. The longest interval between two attacks was 10 months during which period ajmalin was given as an antiarrhythmic drug. During the last months of the patient's life the attacks were particularly frequent, and occurred several times a day. Sometimes the attack was overcome with pethidine or procainamide. However, direct current countershock was mostly used to improve the deteriorating condition of the patient caused by continuous ventricular tachycardia.

It is claimed that the great majority of Adams–Stokes attacks are caused by ventricular tachycardia (Dressler, 1964), and the only promising method to offset this mechanism is the stimulation of the ventricle by an electrical pacemaker (Dressler, 1964). On the basis of experience from treatment of ventricular tachycardias in patients with atrioventricular block, pacing has been attempted against ventricular tachycardia in patients with no such block (Sowton *et al.*, 1964; Härtel *et al.*, 1966). In paired-pulse pacemaking (Chardack *et al.*, 1964), the rate of ventricular contractions can be slowed down by paired electrical stimulations separated by an interval of 150 to 250 msec. Contraction will only follow after the first stimulus.

In the present case both single pulse and paired pulse pacemaking proved ineffective.

Summary

A case of recurrent ventricular tachycardia is presented. The patient was defibrillated 47 times during two and a half years for ventricular tachycardia. Antiarrhythmic agents failed to terminate attacks, as did single and paired pulse pacing. The problem of how to prevent new attacks after successful conversion is discussed. That this problem has not been solved is illustrated by the case now presented.

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